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Cardiac Diseases and Anxiety Disorders

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1. Introduction

Anxiety disorders are amongst the most common psychiatric disorders in all over the world. Its an emotion that prepares the individual to the environmental changes or helps to create a response to those changes. Also there are psychological symptoms such as distress, excitement and a precognition and fear of suddenly something bad going to happen. Anxiety is a symptom that could be seen in many organic disorder and can accompany almost any psychiatric disorder. The relationship between anxiety and cardiovascular system (CVS), is known since the first studies on individuals the 1870's so-called irritable cardiac diseases. With tachycardia and palpitations as a result of severe fear and anxiety related issues, has become a focus of interest in studies that examine the activity of CVS. Nowadays, the relationship between psychological factors and cardiac disease have been discussed. Because creation potential of the sudden death due to cardiac diseases are more sensitive to the psychiatric disorders and development of any cardiac disease might start serious mental issues. These situations are the main subjects of psychocardiology. In addition, mental disorders which has similar symptoms of cardiac diseases (panic disorder (PD), generalized anxiety disorder (GAD), post-traumatic stress disorder (PTSD), etc.) is another problem area. Anxious thoughts causes reduced autonomic variability condition which is a result of decrease in vagal tone. The first reaction to stress is muscle weakness and a feeling of heart stopping due to parasympathetic activation. A short time later, the sympathetic system is activated, sweating, palpitation, tremors, rapid and deep breathing begin. When they do challenging activities or concerned there will be cardiovascular variability and falls occur phasic parasympathetic tone. Studies on this subject emphasize cardiac sensitization caused by sympathetic activity. According to this stimulation of central and peripheral adrenergic structures, catecholamine infusion and behavioral stress can cause cardiac sensitivity in both healthy and ischemic heart. Cardiac diseases within the psychiatric views (whether or syndromal levels of disorder matter), surely, should be recognized and addressed. Patients who work under heavy stressful conditions suffers from continuous excreting of catecholamine with the further aggravated cardiac disease. At the same time, anxiety is caused by a decrease in vagal control also increases the susceptibility to coronary cardiac disease. On most cardiac diseases cases, an intense anger and hostile attitude follows the anxiety. Anxiety came out tops as a leading emotional problem for cardiac patients when it unites with other negative emotions. As a result, the importance of early diagnosis and treatment is obvious due to the reason that occurring simultaneously psychiatric views which emerges with cardiac diseases to cause the illness to worsen. Myocardial infarction, hypertension,

congestive heart failure, pacemaker application in patients often accompanied by psychiatric symptoms. In this chapter the relation between cardiac diseases and the anxiety disorder will be discussed.

2. Cardiac diseases

Cardiac disease refers to any condition affecting the ability of the heart and blood vessels to function properly. There are many different kinds of cardiac disease, but they all threaten the circulatory system in one way or another (Baruah, 2010). This is what makes cardiac disease so deadly; a disruption of the blood supply to any part of the body can lead to tissue damage or death, often within a matter of minutes. Some of the major types of cardiac disease include: Coronary heart disease, hypertensive heart disease, myocardial infarction.

Nowadays, cardiac disease has emerged as the leading cause of death worldwide, particularly in developed countries. The World Health Organization (WHO) reported that 16.7 million deaths in 2003 were caused by some form of cardiovascular disease (Ahrens et al. 2010). Though the rate of cardiac disease is highest in developed countries, developing countries are seeing an increase in the occurrence of cardiac disease (Dakheel et al. 2011). The World Health Organization estimates that by 2010, cardiac disease will surpass AIDS as the leading cause of death in developing countries (WHO 2006).

2.1 Coronary heart disease (CHD)

CHD is the condition that results from the buildup of plaques within the walls of arteries that supply the heart muscle with oxygen and nutrients. CHD can lead to *heart attacks*, which occur when the buildup suddenly interrupts the heart's supply of blood, leading to the damage or death of heart tissue. This is the single largest cause of death in the world. (Furuya et al. 2001, Ko et al.2006, Ingelsson et al. 2007, Baruah, 2010).

2.2 Hypertensive heart disease (HHD)

Hypertension, also known as high blood pressure, can increase the risk for a number of other heart-related conditions, including heart attack, stroke, cardiac arrest, and congestive heart failure (Franklin et al.2011, Simões et al. 2011). In other words, hypertensive heart disease is the target organ response to arterial hypertension. Left ventricular hypertrophy represents an important predictor for cardiovascular events. Myocardial fibrosis, a common end point in hypertensive heart disease, has been linked to the development of left ventricular hypertrophy and diastolic dysfunction (Janardhanan & Kramer, 2011).

2.3 Myocardial infarction (MI)

The damage or death of an area of the heart muscle (myocardium) resulting from a blocked blood supply to the area. The affected tissue dies, injuring the heart. Symptoms include prolonged, intensive chest pain and a decrease in blood pressure that often causes shock (Gul et al.2011). About one quarter of people having an acute myocardial infarction (MI) in the USA will die of it, half of them within 1 hour of the onset of symptoms. Cardiogenic shock develops in over 5% of people surviving the first hour after an acute MI, with a mortality of 50% to 80% in the first 48 hours (Wakai 2011).

3. What is psychogenic cardiac disease?

The pathway toward the current recognition that mental stress and psychiatric illness is a cause of cardiovascular disease has been long. The adverse cardiovascular consequences of panic disorder and chronic mental stress are probable but do remain contentious. A common theme of this field is the importance of neural mechanisms, particularly those involving the sympathetic nervous system, in the origins of cardiovascular disease attributable to stress and psychiatric illness. Evidence exists affirming the mechanistic importance of extreme sympathetic nervous system activation in stress (takotsubo) cardiomyopathy of acute activation of the cardiac sympathetic outflow (Esler 2010). Takotsubo cardiomyopathy (TC) is a neurocardiological disorder presumed to be triggered by stress, which may cause reversible heart failure, usually in postmenopausal women. TC, more recently called transient left ventricular apical ballooning syndrome, is a recently described acute cardiac syndrome. The syndrome is characterised by a sudden onset of transient extensive akinesia of the left ventricle, often involving all three major coronary artery territories, in the absence of significant coronary artery stenosis. The syndrome is accompanied by angina-like chest pain, electrocardiographic changes and minimal release of cardiac enzymes and biomarker levels, mimicking an acute myocardial infarction and is often preceded by an episode of emotional or physical stress, which may play a key role in the pathogenesis of the disorder. The ECG changes are suggestive of an acute coronary syndrome with T-wave inversion with/without ST elevation, most often in the precordial leads. However, the exact mechanism still remains unknown (Sansen & Holvoet 2007). Characteristically, there is only a limited release of cardiac enzymes disproportionate to the extent of regional wall motion abnormality. Transient right ventricular dysfunction may occur and is associated with more complications, longer hospitalisation and worse left ventricular systolic dysfunction. Serial echocardiography is useful to document improvement in cardiac function. Transient mid-cavity obstruction has been invoked with subsequent myocardial stunning in the akinetic segments. Most clinicians are unfamiliar with this disorder. Therefore, some TCs are misdiagnosed as acute myocardial infarction. The modified Mayo Clinic criteria usually confirm a diagnosis, although the diagnostic criteria for TC remain controversial. Enhanced awareness by clinicians is important when encountering patients with chest pain and elevated cardiac enzymes. Treatment is supportive. The most effective long-term management remains to be defined. Although the prognosis is good with recovery of ventricular function at about three weeks, some patients have died (Middlemost & Mabin 2008, Nussinovitch et al. 2011). Complete recovery usually occurs after dramatic presentation, frequently complicated with acute heart failure. Therapy is empiric and directed towards supportive measures against cardiogenic shock, acute heart failure, dysrhythmias. In-hospital mortality rate is less than 1%, but long-term prognosis is still unknown (Putniković et al. 2010). Delineating these biological mediators of heart risk in acute mental stress has provided a potential target for pharmaceutical prevention, to inhibit platelet activation and block adrenergic cardiovascular stimulation. Agreement has been reached that the triggering of myocardial infarction and sudden death by acute mental stress is no longer a hypothetical construct only. It is now proven, is a matter of relevance to the health of the general community, and has led to preventive medical advice being incorporated into national health advisory documents (Esler 2010).

4. Anxiety disorders

Anxiety disorders are a universal phenomenon: exaggerations of evolutionarily hard-wired reactions to dangers to the body or to the psyche. Physicians in earlier times focused on the somatic manifestations of anxiety, such as palpitation, shortness of breath, and the like. Even as recently as the late nineteenth century, the various disorders subsume under the heading of anxiety were described and considered as separate entities, not yet unified as elements of particular class of disorders (Stone 2010). Today, anxiety disorders are classified by DSM-IV-R into four major categories: panic disorder with and without agoraphobia, social phobia and other phobias (eg, simple phobia and agoraphobia without a history of panic attacks), generalized anxiety disorder, posttraumatic stress disorder and obsessive compulsive disorder (Brown & Leyfer 2010).

5. Cardiac diseases and anxiety disorders

A substantial literature supports clinically important associations between psychiatric illness and chronic medical conditions. Patients with severe mental disorders have about twice the prevalence of the classic risk factors for coronary heart disease (CHD) (Birkenaes et al. 2006). Evidence from methodologically rigorous studies of a strong association between CHD and depressive disorders is especially compelling (Shapiro 2005). The prevalence of major depression in patients with CHD is much higher, especially after myocardial infarction, than in the general population. Various psychiatric symptoms are observed on the patients who had myocardial infarction (MI) on their pre, during and after treatment time periods. Also on the treatment period, anxiety, depression, hostile behaviours, aggression, denial, hardships on following medical suggestions, refusing, and as the most severe result, delirium can be observed. Anxiety, is a clinical appearance which is observed on the MI patients during the first 24 hours. With the patient's acceptance to coroner intensive care unit, the patients can show anxious suspense and with this perspective it is possible for patients to see this sickness and it's conditions as a disaster or a treat for themselves. Uncertainty cause anxiety on all patients. Sudden fear of death, the lack of autonomy, deficiency on sexuality, the change of roles on family relationships or losing their status, the fear of having a new infarction risk can cause anxiety on patients. The depression is the most frequently observed symptoms on the patients after their discharge from hospital. Depression also causes delay on the patient's psychosocial recovery. On the other hand, anxiety is frequently observed on patients after their discharge from hospital. This situation prevents the the patients to gain their functionality again on lots of their roles, mainly their sexual life (Hackett et al.1998). Most research focuses on depression, finding that depression can adversely affect self-care and increase the risk of incident cardiac diseases, complications and mortality. Anxiety is also extremely common in patients with CHD. Anxiety disorders are less well studied, but robust epidemiological and clinical evidence shows that anxiety disorders play an equally important role. Biological theories of the interactions between anxiety and cardiac diseases and chronic pain are presented. Available data suggest that anxiety disorders in medically ill patients should not be ignored and could be considered conjointly with depression when developing strategies for screening and intervention, particularly in primary care. Emerging data offer a strong argument for the role of anxiety in medical illness and suggest that anxiety disorders rival depression in terms of risk, comorbidity and outcome (Roy-Byrne et al. 2008). Several studies have suggested an

increased risk of fatal CHD among patients with panic disorder, phobic anxiety and other anxiety disorders. The postulated mechanisms through which anxiety may increase the risk of fatal CHD include hyperventilation during an acute attack, which could in turn induce coronary spasm, or an acute attack of anxiety triggering an episode of fatal ventricular arrhythmias. Nonetheless, it is possible that some unmeasured confounding factors, such as diet or physical exercise, could explain the observed association. On the other hand, the specificity of the association between anxiety and sudden death makes confounding an unlikely explanation for the observed effect. If confounding by diet or exercise could explain the association, one would have expected a positive relationship of anxiety to nonfatal myocardial infarction, as well as to fatal CHD (Kawachi et al.1994). In patients with congestive heart failure, tissue nutrition disrupts due to inadequate pumping ability of heart and in this context some changes might occur in the brain. Symptoms similar to generalised anxiety disorder can be seen, such as breathing difficulties, fatigue, attention and memory deficits as well as expectation anxiety. Anxiety increases the heart rate and blood pressure which results exacerbation in myocard ischemia and worsens the heart failure. And if severity of heart failure increases anxiety increases too (Majani et al.1999, Grubb et al.2000, Aydemir 2006). In patients with chronic stress, constant catecholamine decharge worsens heart diseases even more. Also anxiety causes reduction in vagal tone and that leads to a predisposition at coroner heart diseases. Usually, in patients with heart failure, rage and hostility accompanies with anxiety. Anxiety is one of the leading emotional problems that needs to be dealt with especially if it is together with the other negative emotions. Particularly, anxiety that follows myocard infarction results as more complications and worsens the prognosis (Watkins et al., Moser & Dracup, Aydemir 2006). There are significant associations between anxiety and CHD risk, with a potential biologic link between anxiety and elevations in a biomarker with powerful prognostic factor, namely C-reactive protein (CRP). Previous studies underlined an association between CRP and depression in otherwise healthy adults as well as in CHD patients, potentially indicating a biological link between depression and cardiovascular morbidity and mortality. However, previous research seemed to mainly focus on depression and biomarkers in patients with and without CHD, not taking into account anxiety and specific anxiety disorders. It is not clear whether this association is directly causal or relates to other medical processes among patients with heightened anxiety. Several, mostly epidemiological studies underlined the impact of anxiety on CHD incidence and re-occurrence. In particular, generalized anxiety, generalized anxiety disorder (GAD), and phobic anxiety showed significant associations with CHD (Bankier et al.2008, Bankier et al.2009). Among women with suspected myocardial ischemia, the value of depression symptoms for predicting CHD events varied by the severity of comorbid anxiety. These results suggest that the clinical utility of depression measures may be improved by using them in combination with measures of anxiety (Rutledge et al.2009). Anxiety seemed to be an independent risk factor for incident CHD and cardiac mortality (Roest et al.2010). In men, aged 18 to 20 years, anxiety as diagnosed by experts according to ICD-8 criteria independently predicted subsequent CHD events (Janszky et al.2010). Anxiety is associated with elevated serotonin-mediated platelet reactivity in stable CHD patients and symptoms of anxiety show strong, independent correlations with platelet function (Zafar et al.2010). An initial clinical observation that patients with externally located cardiac pacemakers are more distressed and depressed than those with internally sited pacemakers has been confirmed. In Crisp & Stonehill's study a group of patients with cardiac pacemakers scores more highly on anxiety, phobic and

depressive scales of a standardized psychoneurotic diagnostic questionnaire than do 'normal' populations (Crisp & Stonehill 1969). Patients with cardiac diseases undergoing electrophysiological studies, pacemaker implantation, and myocardial revascularization have different levels and prevalence of anxiety, but they do not show differences in the level and prevalence of depression (Carneiro et al.2009).

5.1 Cardiac diseases and panic disorder

From their symptoms, panic disorder, which gets its name from the mythological God Pan, and consists of unexpected panic attack, can be confused with myocardial infarction. In other words, panic disorder and myocardial infarction can have the same symptom patterns. Regarding to cardiac diseases, panic disorder is the most mentioned anxiety disorder. On major numbers of patients whom suspected to have coronary artery disease, were diagnosed panic disorder with further inspections. On the case of young age group patients complaining especially about chest pain, it is essential to think this as a significant symptom of panic disorder (Halperin 1996). Panic disorder is associated prospectively with coronary artery disease, but the risk of acute myocardial infarction associated with panic disorder has not been specifically investigated. Panic disorder which characterized with the increased oscillation on sympathetic nervous system, increase the risk of cardiac disease due to noradrenergic system dysregulation on locus coeruleus. It was identified as an independent risk factor for subsequent acute myocardial infarction. Comprehensive multidisciplinary approaches are needed to optimize primary and secondary prevention of acute myocardial infarction among patients with panic disorder (Chen et al. 2009). A possible association between the level of anxiety illness severity and sympathovagal balance, which may imply greater cardiac risk. Panic disorder (PD) patients have a heightened or deregulated autonomic nervous system at rest and during autonomic challenge compared with healthy controls (HC) (Martinez et al.2010). Multifiber sympathetic nerve recording has documented massive stimulation of the sympathetic nervous system during panic attacks, accompanied by a surge of epinephrine secretion from the adrenal medulla. Sympathetic nervous tone at rest is normal, but the sympathetic nerves of panic disorder sufferers have been demonstrated to release epinephrine as a cotransmitter. This epinephrine in sympathetic nerves of panic disorder sufferers is presumably taken up from plasma during panic attacks or synthesized in situ by the epinephrine-synthesizing enzyme phenylethanolamine methyltransferase, which has been shown in experimental animals to be induced by chronic mental stress and is present in the sympathetic nerves of patients with panic disorder. This sympathetic nerve epinephrine cotransmission is potentially a cause of cardiac arrhythmias (Esler 2010). Panic disorder has been associated with both an increased risk of coronary events. Hemoconcentration, with both a decrease in plasma volume and an increase in plasma viscosity, is a possible contributor to the risk of acute ischemic events. The acute hemoconcentration observed in relation to pentagastrin-induced panic symptoms may be relevant to the increased risk of stroke and acute coronary events found in patients with panic disorder (Le Melleo et al.2011). In patients with complicated hypertension, panic attack and anxiety has been seen more frequently (Aydemir 2006). Frequency of panic disorders in hypertension patients was reported as 13% (Davies et al.1999).

5.2 Cardiac diseases and post-traumatic stress disorder (PTSD)

PTSD occurs after traumatic life events, appears under circumstances reminds situations like facing death or death related events and characterized by couple of symptoms such as

avoiding behaviour, living the event in an unpleasant way over and over again during a dream or in memory and accompanied with severe anxiety. Especially in patients who recognizes MI as a trauma, often PTSD can be observed. Whenever patient perceives a heart related stimulant he goes back to the beginning and live the event all over again (Aydemir 2006). PTSD makes MI hard to be dealt with for the patient and physiologically it might delay trombolysis (Alonzo 1999).

6. Treatment

Psychiatric disorders are common feature of heart disease patients and possibly stem from their common biochemical background. Depression, anxiety and heart failure co-morbidity has several clinical implications on the prognosis of these patients. Furthermore antidepressant drugs have known cardiovascular side effects, while their safety and efficacy in heart failure has not been fully elucidated yet. The right choice of antidepressant treatment in cardiac diseases an issue of high importance as it can affect the clinical outcome of these patients. Even though certain conclusions cannot be drawn yet, evidence suggests that the use of selective serotonin reuptake inhibitors may have a beneficial effect on clinical outcome of heart failure patients (Tousoulis et al.2010). Tricyclic antidepressants and SSRIs may be associated with increased risk of mortality, and SSRIs with increased risk of hemorrhagic and fatal stroke, although absolute event risks are low (Smoller et al.2009). The quinidine-like effects of some antidepressant drugs (particularly tricyclic antidepressants) and depression in patients with major mental illness. The tricyclic antidepressants (TCAs) have various effects on the cardiovascular system, including Type IA antiarrhythmic activity that has been associated with an increased risk of mortality in post-myocardial infarction patients. This is especially true among elderly patients with existing risk factors for corrected QT (QTc) interval prolongation. Vieweg et al. (2009) used PubMed, previously reported review articles and the extensive personal files of the authors to identify cases of subjects aged ≥ 60 years who developed QTc interval prolongation, polymorphic ventricular tachycardia (PVT)/torsade de pointes (TdP) and/or sudden cardiac death while taking antipsychotic or antidepressant drugs or a combination of these medications. They identified 37 patients who had taken, in total, 46 antipsychotic or antidepressant drugs. Their most striking finding was that almost four-fifths of their cases involved women. When the 14 critically ill subjects receiving haloperidol intravenously were excluded, 91.3% of their subjects were women. Almost three-quarters of their study subjects had cardiovascular disease. Specifically, such clinicians treating elderly patients with antipsychotic and antidepressant drugs that may prolong the QTc interval should aggressively obtain a baseline ECG for elderly female patients with additional risk factors such as personal or family history of pre-syncope or syncope, electrolyte disturbances or cardiovascular disease. Elderly male patients are also subject to QTc interval prolongation when such risk factors are present. It is important that the clinicians themselves inspect ECGs. If the QT interval is more than half the RR interval, QTc interval prolongation is likely to be present (Vieweg et al. 2009). Some antidepressant agents can cause electrophysiological changes of cardiac function leading to ventricular arrhythmias and sudden death. However, antidepressants have also protective effects on the heart through their capacity to modulate cardiac autonomic-mediated physiological responses. Heart rate variability and QTc length are two strictly linked parameters that allow us to appreciate the effects of different drugs on cardiac physiology. Heart rate variability reflects functioning of the autonomic nervous system and

possibly also regulation by the limbic system. Autonomic regulation of cardiac activity influences also cardiac repolarization and QT length, both directly and via its effects on heart rate (Sala et al. 2009). There is a significant body of evidence suggesting that the presence of depression is independently associated with a decline in health status and an increase in the risk of hospitalization and death for patients with coronary artery disease or congestive heart failure. Novel treatment modalities such as selective serotonin re-uptake inhibitors (SSRIs) may improve depressive symptoms, anxiety symptoms and prognosis of post-myocardial infarction and heart failure patients interacting with the common pathophysiologic mechanisms of depression and cardiovascular disease (Paraskevaidis et al. 2006). SSRIs are established agents for the treatment of depression and are well tolerated in patients with cardiac disease. SSRIs are a heterogeneous group of antidepressants, which apart from their common mechanism of action, differ substantially in their chemical structure, metabolism and pharmacokinetics. Intervention with sertraline has the potential to provide depressed patients with cardiac disease relief from their depressive symptoms, improvement in quality of life and a potential benefit in their cardiovascular risk profile (Parissis et al. 2007). But, escitalopram overdose leading to prolongation of the QTc interval has only twice been previously described in the literature. In the event of an overdose, QT prolongation can occur and ECG monitoring should take place for at least 2 days after ingestion in order to prevent life-threatening arrhythmias such as torsades de pointes (tdp). Other factors and drugs that could contribute to prolongation of the QT interval should be taken into account when determining the time period needed for ECG monitoring in the individual patient (Mohammed et al. 2010). Tianeptine therapy for affective disorders concurrent with MI causes an evident reduction in psychopathological symptomatology and a statistically significant decrease in Hamilton Depression Rating Scale and Hamilton Anxiety Rating Scale scores (Vasiuk et al. 2010). There is some evidence for the safe and at least modestly effective use of psychotherapy and antidepressants to treat depression and anxiety disorder in heart failure patients. Cognitive behavioral psychotherapy and selective serotonin reuptake inhibitors are first line treatments. The efficacy of depression treatment in altering cardiac outcomes in heart failure patients has yet to be established (Shapiro 2009). The SSRI paroxetine was compared with a therapeutic level of the TCA nortriptyline in a randomized, controlled study and demonstrated a benign cardiovascular profile, while the TCA induced a significantly higher rate of serious adverse cardiovascular events. On the basis of this favorable cardiovascular profile, the SSRIs should therefore be the preferred choice for the treatment of most patients with comorbid depression and cardiovascular disease. Investigation of putative pathophysiologic mechanisms linking depression and cardiovascular mortality, such as the role of platelet activation, will form the basis for further investigation of antidepressant treatments in order to establish if the antidepressants have a beneficial effect on the prognosis of cardiovascular diseases (Roose 2001).

7. Conclusion

Anxiety disorders those occur along with a cardiac disease must be recognised in early stages and must be treated with care. The effects of the drugs over heart and drug-drug interactions must taken into account of medical treatment. If we apply all these principles, the results would be pleasing. During the treatment clinicians must be sensitive about their patient's psychiatric symptoms and disorders.

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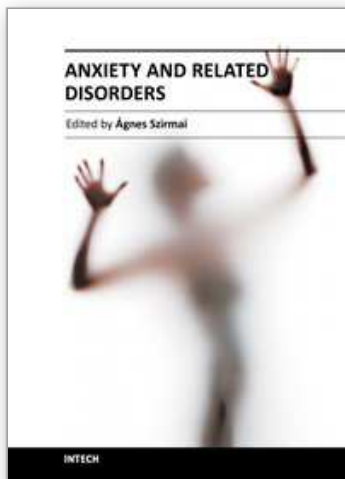
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Anxiety disorders are one of the most common psychiatric disorders worldwide and many aspects of anxiety can be observed. Anxious patients often consult primary care physicians for their treatment, but in most cases they do not accept the diagnosis of anxiety disorder. Anxiety is a symptom that could be seen in many organic disorders and can accompany almost any psychiatric disorder. Anxiety disorders are frequent and are associated with significant distress and dysfunction. Stigmatization is an important factor in insufficient diagnosis. The problems of anxiety cover all fields of life. This book intends to describe the epidemiological aspects and the main co-morbidities and consecutive diseases of the anxiety disorders.

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